A Review of the Role of Chronic Traumatic Encephalopathy in Criminal Court

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Chronic traumatic encephalopathy (CTE) is believed to be a degenerative brain disease characterized by repetitive brain trauma resulting in a specific pattern of neuropathological changes, which some have linked to functional disturbance and aggression. The diagnosis has gained greater public attention after these same neuropathological changes were discovered in multiple deceased National Football League (NFL) players, many of whom had exhibited signs of aggression, impulsivity, and poor executive functioning, according to a widely publicized study. When an NFL player convicted of murder was found to have the neuropathological changes associated with CTE following his suicide, the *New York Times* editorial section asked whether CTE was a defense for murder. This idea raises an interesting legal and philosophical question about whether an individual's criminal actions can be determined by something outside their control, such as past head trauma. To begin to attempt an answer, this article reviews what is currently known about the neurobiology of traumatic brain injury, CTE, and morality. By looking at how U.S. criminal law courts have handled cases of dementia and traumatic brain injury in the past, we can better understand how to consider this postmortem diagnosis in its forensic context.

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In 2017, Aaron Hernandez, a star player for the National Football League, died by suicide in prison where he was serving a life sentence for first-degree murder.[1] On autopsy, Mr. Hernandez's brain exhibited neuropathological changes consistent with the proposed criteria for a diagnosis of chronic traumatic encephalopathy (CTE), a neurodegenerative disease thought to be associated with repetitive brain trauma and behavioral changes such as aggression, dementia, impulsivity, and poor executive functioning.[2] Similar conditions have been described over the past century. Initially termed "dementia pugilistica," and described as "punch drunk," the diagnostic predecessor to CTE was thought to be unique to boxers sustaining forceful blows to the head and repeated episodes of loss of consciousness.[3,4] Renewed interest in the disease emerged in the 2000s, following the publication of several widely publicized studies by the laboratories of Ann McKee and Bennet Omalu, who linked consistent neuropathological changes to behavioral phenotypes in U.S. athletes, particularly football players.[2,5,6] More newspaper headlines came in July 2017 when McKee's group studied the donated

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brains of 202 deceased football players with neurocognitive, emotional, and behavioral problems prior to death and reported that 177 met the recently established neuropathological criteria for the relatively new diagnosis of CTE.[7]

Such findings led the *New York Times* editorial page to ask, "Is CTE a Defense for Murder?"[8] The authors, who are also law professors, answered plainly, "Mr. Hernandez should not have been convicted of first-degree murder. Given the conclusive diagnosis of Stage 3 CTE, it is likely that a lifetime of playing footbal —not Mr. Hernandez's will—was to blame."[8] A greater understanding of the science behind this diagnosis and the legal literature, however, suggests that the answer is not nearly so simple.

Defining CTE

CTE is a diagnosis that has been in the literature for the better part of a century. Originally recognized in boxers, case report data led early physicians to believe the clinical features of this illness, thought to be a result of numerous blows to the head, were primarily gait disturbance, dysarthria, tremor, and cognitive impairment.[9] Over time, the name of the disorder, definitions, and clinical findings have changed, as further case reports and small observational studies were added to the literature. The consensus has been that CTE is due to the cumulative impact of numerous subconcussions and concussions, or mild traumatic brain injuries.[10] As defined by the Department of Veterans Affairs and the Department of Defense, mild traumatic brain injuries are head injuries with a loss of consciousness of less than 30 minutes, altered mental status of less than 24 hours, and no findings on neuroimaging.[10] Individuals with concussions do tend to have some symptoms (including headache and confusion) though they tend to be brief and self-limited.[10] Subconcussions occur when a blow to the head occurs with no appreciable postinjury symptomatology. The vast majority of traumatic brain injuries sustained by all individuals are subconcussions or mild traumatic brain injuries, but athletes, particularly football players, sustain exponentially more subconcussions and mild traumatic brain injuries than the average individual.[12] Effects from such cumulative brain traumas are thought to cause a pattern of neuropathological changes called CTE.[13] CTE can only be diagnosed postmortem via a constellation of neuropathological changes. Several CTE researchers, including Omalu, have published case reports of individuals with CTE, diagnosed postmortem. He believes that CTE findings correlate with collateral reported history of memory loss, headache, executive dysfunction, language difficulties, aggression, apathy, motor disturbance, and dementia (Table 1).[11]

Table 1 Symptoms Associated With Chronic Traumatic Encephalopathy

Behavioral	Cognitive	Motor
Depression[2,11]	Executive dysfunction[2]	Dysarthria[11]
Aggression[2]	Word-finding difficulties[2]	Unstable gait[11]
Poor financial decisions[11]	Concentration challenges[2]	Parkinsonism[2]
Suicidality[2,11]	Attention difficulties[2]	
Tendency toward substance use[11]		

There are several neuropathological changes that are described as characteristic of CTE. These include depositions of hyperphosphorylated tau (p-tau) protein aggregates in neuronal and astrocytic cells in the brain.[14] Distinguishing it from other tauopathies like Alzheimer disease, CTE p-tau aggregates are uniquely distributed in perivascular spaces, at the depths of the cortical sulci, in an irregular pattern.[5,14] Similar to other tauopathies, CTE is also associated with the accumulation of amyloid plaques, and p-tau aggregates in cortical layers II and III of hippocampal regions CA2 and CA4.[16] Macroscopically, researchers report seeing frontotemporal atrophy, an overall reduction in brain mass, and increased ventricle size, as well as a cavum septum pellucidum.[5] These macroscopic changes are associated with disinhibited behavior and cognitive impairment in other neurodegenerative illnesses as well, such as Alzheimer's disease and frontotemporal dementia.

McKee's group has postulated that CTE is progressive based on a variance of severity of cases seen postmortem.[2] Omalu's group, however, has opined that differences in severity may be due to CTE consisting of several different illness groups. In each case, the illness itself is relatively stable, but the severity may differ by the subtype of CTE based on neurofibrillary tangle distributions.[16] These distributions change based on the number and type of head injuries endured. There are further differences among studies. For example, Omalu states there is no atrophy in CTE,[6] whereas McKee says widespread atrophy is common.[2] McKee's group feels that p-tau astrocytic tangles are pathognomonic for the disease,[2] whereas Omalu believes these are not

present in all cases.[16] The National Institutes of Health has developed a clinical consensus as to what pathological features must be present for a diagnosis to be reached. Primarily, perivascular tau accumulations in astrocytes and neurons in an irregular pattern deep in the sulci are considered diagnostic; other abnormal findings could be supportive or exclusionary.[14]

The neurocognitive and behavioral changes that some researchers have proposed as being associated with CTE are quite broad and encompass a large spectrum of severity. Researchers have differing opinions as to what must be present for diagnosis. McKee and her group have argued that there is a range of CTE symptoms, from no clinical manifestations[2] to severe CTE consisting of ataxia and mood dysregulation, impulsivity, and cognitive impairments similar to those with advanced dementia.[17] Omalu, on the other hand, believes symptoms must be present for diagnosis.[11]

What is truly known about CTE and its symptomatology is still quite limited because sample sizes have been small and heterogeneous, and research has been conducted largely on postmortem brains. Thus, the majority of behavioral and cognitive correlations to neuropathology must be inferred.[15] Postmortem studies are inherently biased, with samples coming from relatives who might be inclined, *a priori,* to attribute problematic personality traits and behaviors to neuropsychiatric conditions. Prospective studies following patients with head injuries and various exposures to brain trauma and other potentially modifying variables are still needed to establish definitively the causation between repeated brain trauma and CTE.[18] There does appear to be a pattern of p-tau deposition unique to athletes exposed to multiple mild head injuries, but whether this finding has clinical implications, particularly implications that are distinctive from other established forms of dementia and neuroinjury, is still open for debate. Symptoms such as aggression, memory loss, and impulsivity are ultimately nonspecific and are seen in a variety of pathologies and personality types. Given that behavioral correlations are made postmortem, establishing the chronology of symptoms is very difficult. For example, a football player may have been impulsive prior to many of his neuroinjuries and may have had earlier depressive tendencies. Thus, to attribute these characteristics solely to neurotrauma would be inaccurate.

The Putative Neurobiology of Morality

Regardless of whether CTE is a validated diagnosis in and of itself, a larger question looms. If we are to consider CTE as a defense for wrongdoing, we must assume that morality and behavior are at least partially neurobiologically mediated. There is some evidence that this may be the case. One of the most commonly injured areas of the brain in traumatic brain injury (TBI) is the ventromedial prefrontal cortex (VMPFC).[19] The VMPFC serves as an inhibitory control center for the limbic system, the seat of the fight-or-flight response. Thus, its damage results in anxiety, impulsivity, and aggression, representing unmitigated fight-or-flight responses.[19,20] Studies of what are thought to be more advanced cases of CTE have revealed advanced gray and white matter atrophy in multiple areas of the brain, typically most severe in the frontal lobe, specifically in the VMPFC.[13] McKee *et al.*[2] agree that damage to this area may underlie the lack of insight and aggressive tendencies seen in this population.

The results of multiple other neuromodulatory and neuroimaging studies further suggest that the VMPFC may be the structure responsible for one's innate moral sense.[21] Fumagalli *et al.* targeted the VMPFC with transcranial direct current stimulation (tDCS) in control subjects. Responses to moral dilemmas changed significantly in female subjects after tDCS. Anodal tDCS appeared to decrease VMPFC activation, acting like a lesion. After anodal tDCS, female subjects responded with a colder, more detached, and "utilitarian" pattern on moral dilemma.[22] Other disorders associated with aggressive behavior have been reported to involve VMPFC pathology, as would be expected if the VMPFC were the essential structure responsible for moral behavior. For example, about half of individuals with frontotemporal dementia exhibit antisocial (amoral) behavior, and those who exhibit such behavior have clear VMPFC atrophy.[23] Yet another study published by the Fumagalli *et al.* group reported that psychopathic individuals had significantly less VMPFC activation on functional magnetic resonance imaging when shown upsetting, emotionally charged images.[21]

Incarcerated individuals are another candidate population for studying the connection between brain trauma and aggressive, criminal acts. A 2012 meta-analysis estimated that 60 percent of inmates across many studies have a history of TBI, a significantly higher incidence than in the general population.[24] A 2016 cohort study of more than one million young adults living in Ontario, Canada, used government health and incarceration records

to analyze the potential association between TBI and later incarceration. The study reported that individuals with a history of TBI were approximately 2.5 times more likely to be incarcerated than those without prior TBI (2.47 times for men, 2.76 times for women).[25] A 2011 study of 200 Australian prisoners with 200 controls matched by place of residence reported that TBI was associated with impulsivity, dissocial (antisocial) personality traits, alcohol use, illicit drug use, and level of education.[26] Though TBI was significantly more common in the prisoner group, TBI frequency was not significantly associated with incarcerated status, whereas the following factors were: lower education, drug use, alcohol abuse, impulsivity, and dissocial traits. Moreover, when TBI was removed from the logistic model, the odds ratio for incarcerated status was relatively unchanged for impulsivity and dissocial traits. In contrast, when impulsivity and dissocial traits were excluded from the model, a weak association was noted between TBI frequency and incarcerated status.

A Hypothetical CTE Defense

The literature suggests an argument could be made that individuals with CTE might be predisposed to impulsive, aggressive, and antisocial behavior due to damage to putative morality-associated neurobiological circuits, which, if intact, would serve to inhibit such behavior. To address whether CTE could be a defense for murder, as the *New York Times* asked,[8] we must also review how mental health and other medical experts work with the legal system on matters of criminal accountability. The insanity defense and related criminal defenses (e.g., diminished capacity or diminished responsibility) focus on two main elements: the defendant's mental state at the time of the offense and how exactly that state incapacitated the defendant at the time. The incapacities to be investigated vary with jurisdiction and with the asserted defense, but they may include the ability to appreciate the legal or moral wrongfulness of the criminal act charged and the ability to refrain from committing that act. The expertise of mental health and other medical professionals is most relevant and helpful to the question of the defendant's mental state at the time of the offense. Capacity evidence presumes concepts fundamental to criminal law and justice, such as free will and equality of choice, which are potentially at odds with neuroscience, given that the neurobiological bases for these concepts have not been established.

An Expert's Possible Role

A forensic psychiatrist asked to evaluate a living defendant claiming that CTE rendered him insane would face an immediate and likely insurmountable barrier: CTE cannot currently be diagnosed in a living defendant because the diagnosis relies on postmortem studies. Therefore, testimony suggesting that a defendant had CTE that rendered him insane at the time of the offense might well be considered inadmissible from the start based on either a *Frye* test or a *Daubert* test of admissibility.[27]

Setting this aside, an evaluator approaching such a case would first focus on the defendant's medical and psychiatric history, noting other conditions that might mimic or confound the diagnosis. Particular attention would be given to the defendant's history of head injury, including TBI as well as subclinical injuries. A standardized instrument, such as the Ohio State University TBI Identification Method-Interview Form,[28] might be used to identify past head trauma, but more impartial data sources such as hospitalization, school, legal, or military records would be desirable to corroborate the defendant's claims for both the incidence of trauma as well as tracing the onset and progression of behavioral changes. Temporal correlation between the onset of behavioral changes and head trauma, as well as correlation with other comorbid or potentially confounding conditions, would be crucial. The mental status examination would focus on signs of behavioral disinhibition and cognitive deficits. Although there is no standardized clinical instrument for the diagnosis of CTE, neurocognitive testing with validity testing would be recommended in such a case, as would a neurological examination. At the least, without strong correlation between repetitive brain trauma and later onset of behavioral and cognitive changes with consistent mental status examination and neurocognitive testing findings, a CTE diagnosis could not be made, much less any proposition that it affected the defendant's mental state at the time of the offense.

Future Possibilities for CTE Defenses

The possibility of diagnosing CTE without postmortem studies could soon be on the horizon.

Researchers are beginning to use positron emission tomography scans to attempt to diagnose CTE in living subjects, with some promising results.[29] If that science advances, courts could potentially allow a well-reasoned expert's opinion in favor of a CTE defense. In the context of the insanity defense, the first question is whether an individual's abnormal behavior qualifies as a mental disease. So long as the question is deemed a matter of fact, the defense has the opportunity to prove to the finder of fact that the accused is properly diagnosed with the disorder. Next to prove would be whether a given illness sufficiently compromised the defendant's cognitive (whether moral or legal knowledge) or volitional (whether limited to instantaneous impulses or broadened to longer-term control deficits) capacities. The definition and inclusion of such capacity dimensions are dependent on the law in the relevant jurisdiction. A review of similar situations in cases involving frontotemporal dementia indicated that affected individuals may have relatively intact cognitive faculties and are often aware of the wrongfulness of their actions but exhibit deficits in their volitional capacities.[30]

If a full insanity defense is unavailable at the guilt phase, a diminished capacity or diminished responsibility defense might be another avenue for the defense team. Testimony would be given on the defendant's capacity to form intent, and experts could speak of the cognitive and behavioral shortcomings of the defendant and people like the defendant. In terms of outcome, a successful diminished capacity assertion typically leads to conviction on a lesser included charge. An insanity verdict typically results in compulsory mental health treatment until the defendant is no longer deemed dangerous because of mental illness. Overcoming the dangerousness prognosis and stigma is a dubious prospect in cases of neurodegenerative diseases.

A potential complication for the defense team seeking to use a CTE defense during the sentencing phase is that CTE could be taken as proof of dangerousness and thus could be more aggravating than mitigating. Courts have generally been receptive to admitting neuroimaging evidence of alleged brain abnormalities, especially at the sentencing phase in capital cases where the judge or jury must consider all potentially mitigating evidence presented. The evidence may backfire from the defense's perspective, however, if the sentencer, whether articulated or not, counts it in aggravation of the crime.

A famous case comes from Missouri, where in 1974 a sawmill accident left Cecil Clayton with significant brain trauma. It reportedly led to profound personality changes, culminating 22 years later (in 1996) in his killing a sheriffs' deputy during a domestic dispute. Despite his lawyers' raising both diminished capacity and sentence mitigation level defenses based on the history of injury, Mr. Clayton received the death penalty. In the various appeals that followed, the insanity defense was also raised, in addition to other defenses not previously described. In 2015, Mr. Clayton was executed at age 74, the oldest person on Missouri's death row.[31]

Conclusions

Although there is a growing body of evidence that human moral behavior is partly inherited and partly embodied in the VMPFC, an area of the brain often injured in TBI, much stands in the way of using CTE as a defense for murder at this time. First, there is the obvious obstacle of having expert testimony asserting a CTE diagnosis reach a general acceptance standard. The biological validity and reliability of CTE is untested. There is no consensus as to whether CTE is a discrete illness and whether it is distinguishable from other forms of dementia and neurotrauma. Furthermore, we lack an empirical means to determine if the symptoms blamed on the neuropathological findings of CTE are caused by them.[32] Even if CTE is determined to be the entire cause of an individual's problematic behavior, there are examples of other forms of dementia not leading to successful defenses for individuals who have committed crimes.

As of now, the only way to diagnose CTE is postmortem, and that diagnosis itself is still debated. Our current understanding of CTE is rapidly changing, however. Multiple researchers are working on better characterizing the pathological changes associated with the disease in animal models and now in living individuals believed to have the condition. Labs are using positron emission tomography neuroimaging to find biomarkers that may, one day, prove to be pathognomonic for the disorder.[29] Existing studies are hampered by small samples and heterogeneity, but this may change as neuroimaging abilities evolve. Whether neurobiological defenses can more reliably succeed in the future will likely require evolution in jurisprudence in addition to, and hopefully tracking, advances in neuroscience.

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